Article : Basal Metabolic Rate and Metabolic Measurements as Predictors of Insulin Resistance in Females with Different Phenotypes Polycystic Ovarian Syndrome

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ABSTRACT

*Polycystic ovarian syndrome (PCOS) is associated with metabolic complications such as insulin resistance (IR) , visceral obesity, and dyslipidemia contributing to an increased lifetime risk of developing cardiovascular diseases, type 2 diabetes mellitus, and hypertension. Aimed to evaluate the new metabolic indices and basal metabolic rate to predict insulin resistance in polycystic ovary syndrome phenotypes. Method**s** This study as a case-control study and 210 females including 140 female cases effect by different phenotypes of PCOS and 70 healthy females, the metabolic indices were determined waist /hips ratio(WHR) ,body mass index(BMI), lipid accumulation product (LAP), basal metabolic rate (BMR), and body adiposity index (BAI) and visceral adiposity index (VAI), serum hormonal levels and insulin concentration were determined by the electrochemiluminescence immunoassay "ECLIA" system (Cobas e 411, Roche Diagnostic, Germany) . Using the ROC curve, the accuracy of the prediction value was determined.The result, in this study was that all PCOS phenotypes significant increase compared to the control group with a p-value < 0.05 , interestingly, phenotype A was shown high level in VAI , BAI,LAP and BMR . Receiver Operator Characteristic Curves(ROC) analysis for VAI and BMR showed good diagnostic performers to word phenotype A. ROC for LAP and BAI were the best predictions for phenotypes group C. Conclusion, The females with phenotype A have a higher basal metabolic rate and metabolic measurements than the control group. Consequently, insulin resistance and metabolic disturbances more severe in phenotype A than in other phenotypes.*

Keywords: *Polycystic Ovarian Syndrome Phenotypes, metabolic indices, basal metabolic rate, adiposity, insulin resistance.*

الخلاصة

الخلفية: متلازمة تكيس المبايض (PCOS) مرتبطة بمضاعفات التمثيل الغذائي مثل مقاومة الأنسولين (IR) والسمنة الحشوية وخلل شحميات الدم مما يساهم في زيادة خطر الإصابة بأمراض القلب والأوعية الدموية ومرض السكري من النوع 2 وارتفاع ضغط الدم.الهدف من الدراسة إلى تقييم مؤشرات الأيض الجديدة ومعدل الأيض الأساسي للتنبؤ بمقاومة الأنسولين في الأنماط الظاهرية لمتلازمة تكيس المبايض .طرق العمل هذه الدراسة كدراسة حالات وشواهد و 210 إناث بما في ذلك 140 حالة أنثوية أثرت بواسطة أنماط ظاهرية مختلفة من متلازمة تكيس المبايض و 70 أنثى سليمة ، تم تحديد مؤشرات الأيض نسبة الخصر / الوركين (WHR) ، مؤشر كتلة الجسم (BMI) ، منتج تراكم الدهون تم تحديد (LAP) ، ومعدل الأيض الأساسي (BMR) ،

ومؤشر السمنة في الجسم (BAI) ، ومؤشر السمنة الحشوية (VAI) ، ومستويات الهرمونات في الدم وتركيز الأنسولين بواسطة نظام المقياس المناعية "ECLIA" اللمعان الكهربائي (Cobas e 411، Roche Diagnostic ، ألمانيا). باستخدام منحنى ROC ، تم تحديد دقة قيمة التنبؤ.النتائج في هذه الدراسة ، كانت جميع الأنماط الظاهرية لمتلازمة تكيس المبايض تزداد معنوية مقارنةً بالمجموعة الضابطة بقيمة p <0.05 ، ومن المثير للاهتمام أن النمط الظاهري A أظهر مستوى مرتفعًا في VAI و BAI و LAP و BMR. أظهر تحليل المنحنيات المميزة لمشغل المستقبل (ROC) لـ VAI و BMR أن الأداء التشخيصي الجيد للنمط الظاهري للكلمة A. ROC لـ LAP و BAI كانت أفضل تنبؤات لمجموعة الأنماط الظاهرية C.

الاستنتاج ، فإن الإناث ذوات النمط الظاهري (A) لديهن معدل استقلاب أساسي وقياسات أيضية أعلى من المجموعة التحكم. وبالتالي ، قد تكون الاضطرابات الأيضية أكثر حدة في النمط الظاهري (A)منها في الطرز المظهرية الأخرى .

INTRODUCTION

Polycystic ovarian syndrome (PCOS) is the most common endocrine condition in females of reproductive age , with a global prevalence of 4 to 20% depending on the diagnostic criteria used to define the syndrome **(1).** PCOS appears to represent more than one disease due to its widespread occurrence. Ovarian androgen production rises when the ovary has trouble converting androgen to estrogen. The aromatase in adipose tissue converts these androgens into estrogen, It prevents the release of FSH while encouraging the production of LH, leading to the elevated LH and LH/FSH ratio characteristic of a syndrome **(2).**

Because of heightened LH stimulation, many ovarian follicles stop developing between the preantral and antral phases because to arrest in the theca cells , leading to follicular fluid accumulation generating cyst-like formations along the periphery of the ovary (theca cell hyperplasia) , making it look like a string-of-pearls . **(3).** Due to an increase in the number of follicles and the expression of key enzymes involved in androgen synthesis, an excessive quantity of androgens is produced. As a result of PCOS, a rising in the frequency of insulin resistance (IR) and obesity worldwide **(4)**. The Rotterdam -criteria, in 2012 this criterion required two out of the three following features to occur (after the exclusion of related disorders): ovulatory dysfunction (oligo/ amenorrhea) (OD), clinical and/or biochemical hyperandrogenism (AH), or polycystic ovaries morphology (PCOM) ,it has four phenotypes A(AH, OD, and PCOM), phenotype B(AH and OD), phenotype C(AH and PCOM) and phenotype D(OD and PCOM) **(5).** PCOS is associated with metabolic aberrations such as insulin resistance (IR) , visceral obesity, and dyslipidemia contributing to an increased lifetime risk of developing cardiovascular diseases, type 2 diabetes mellitus, and hypertension (**6**) . IR and compensatory hyperinsulinemia are believed to play a crucial role in the pathogenesis of PCOS and are associated with a number of its phenotypic characteristics. Different explanations have been suggested to explain the development of insulin resistance in PCOS**(7).** Obesity is highly linked to PCOS and is related to metabolic problems as well. It's a typical symptom for females with PCOS. Half of PCOS patients are overweight or obese (**8**). It has been demonstrated that PCOS phenotypes are associated with different risks, Hyperandrogenemia, obesity, and severe menstrual irregularities are dependent predictors of metabolic abnormalities in PCOS **(9).** According to the research, patients with PCOS phenotypes A and B have more severe menstrual abnormalities, Hyperinsulinemia (HI) and IR are hallmarks of this phenotype, and patients with this phenotype are at a far higher risk of developing metabolic syndrome than phenotype D individuals **(10).** Phenotype A patients are the most likely to be obese (86.0%), followed by phenotype B (27.9%), phenotype C (46.6%), and phenotype D (38.8%) **(11).** Most studies found that patients with nonandrogenic phenotype D in PCOS do not have significant endocrine and metabolic disorders; consequently, the prevalence of the metabolic syndrome is low **(12).** The metabolic disorders of PCOS are mainly related to hyperandrogenism and compensatory hyperinsulinemia and occur dependently on obesity**(13).**

The magnetic resonance imaging, ultrasonography, dual X-ray absorptiometry and Bioelectric Impedance Analysis (BIA) are various methods used to show obesity. However, more methods it needs to evaluate visceral adiposity because these methods cost and are complex to step into these devices. Therefore, new metabolic measurements such as visceral adiposity index (VAI), lipid accumulation product (LAP), basal metabolic rate (BMR), and body adiposity index (BAI) have been considered in recent research (**14-15**).

The aimed to evaluate the new metabolic indices and basal metabolic rate to predict insulin resistance in polycystic ovary syndrome phenotypes

MATERIAL AND METHOD

A. STUDY POPULATION

The current case-control study of 210 females included 140 females with PCOS divided into four phenotypes (A, B, C and D), and 70 control women. Patients with PCOS were gathered from the outpatient clinic and reproductive fertility consultant at the Teaching Hospital of Obstetrics and Gynecology in the Kerbala health directorate in Iraq**.** Each woman in the study was given a thorough interview that included questions about her background, her family's background, her demographics, and a clinical assessment in the lab.The Rotterdam criteria from 2012 were used to get the PCOS diagnosis.Clinical and/or biochemical hyperandrogenism, oligo/anovulation, and ultrasound evidence of polycystic ovaries (>12 follicles 2-9 mm in diameter or ovarian volume >10 mL in at least one ovary) were considered diagnostic of polycystic ovary syndrome (**16**) , thorough physical was performed on each and every woman. The modified Ferriman-Gallwey scoring system was used to determine whether a person has hirsutism. Nine different body parts [the upper lip, chin, chest, upper and lower abdomen, thighs, upper and lower back, and upper arms] are scored from 0 to 4, a score of 0 represents the absence of terminal hair growth, and a score 4 represents extensive growth If a patient scores four or more points based on the cut-off race-specific modified Ferriman-Gallwey scale, they are considered to have clinical hyperandrogenism-hirsutism (**17**). The presence of acne and male-pattern baldness was assessed. Oligo/amenorrhea was defined as having fewer than six menstrual cycles per year. Ovarian volume greater than 10 ml on ultrasonography and fewer than 12 follicles measuring 2 to 9 mm are indicative of polycystic ovarian morphology (PCOM).

Criteria for exclusion Women with a history of taking any other medication (lipid-lowering agents, contraceptive pills, ovulation stimulation, corticosteroids, antidiabetic, and antihypertensive medications), as were women with autoimmune disease, T2DM, thyroid disease, hyperprolactin, cardiovascular disease, hypertension, chronic liver failure, chronic renal failure, and malignant diseases.

B. CALCULATIONS AND LABORATORY MEASUREMENTS

Serum follicle-stimulating hormone (FSH), luteinizing hormone (LH), prolactin, thyroid-stimulating hormones (TSH), and insulin concentration were determined by the electrochemiluminescence immunoassay “ECLIA” system (Cobas e 411, Roche Diagnostic, Germany). Fasting blood glucose was determined by a Clinical chemistry analyzer(Monarch 240, Biorex Diagnostic, United Kingdom).serum lipid concentration ((total cholesterol (TC), high-density lipoprotein cholesterol (HDL-C), low-density lipoprotein (LDL)and triglycerides (TG)) fully automatic chemistry analyzer(SMART-120, Geno TEK, United States of America).serum free testosterone hormone fully -auto chemiluminescence immunoassay analyzer((MAGLUMI 600, Snibe Diagnostic, Germany). Morning samples of fasting blood were collected between the second and fourth day of the cycle. The formula for calculating body mass index (BMI) is body weight in kilograms divided by height in meters squared (kg/m2). For women with abdominal obesity, waist circumference was measured in the standing position, and WHR was calculated as the ratio of waist (cm)to hips (cm). Homeostasis model assessment of insulin resistance (HOMA-IR) was calculated as [fasting sugar (mg/dl) x fasting insulin (μU/ml) /405] (**18**). The Mifflin-St Jeor for Basal Metabolic Rate (BMR) has used the following equation:

BMR (Female):[(10 × weight in kg) + (6.25 × height in cm) - (5 × age in years) – 161] (**19**). Visceral obesity is investigated using the formula VAI, LAP, and BAI in (Table (1))

Table (1): Formula metabolic measurements

Metabolic measurements
BAI %= hip circumference (cm)/height (m)$^{1.5}$ – 18 (20)
VAI (Female) = [WC (cm)/ (36.58+ (1.89 × BMI)] × [Triglycerides (mmol/l)/0.81]× [1.52/HDL (mmol/l)] (21)
LAP (Female) = (WC in cm – 58) × TG in mmol/L (22)

C. STATISTICAL ANALYSIS

The baseline data will descriptive be Mean± standard deviation values were used for parametric variables. For analysis of all data used Kruskal Wallis test with a P value of 0.05 will consider statistically significant. Multinominal logistic regression was performed to analyze the association between the analyzed factors was estimated using odds ratios (ORs) and a 95% Confidence Interval Range which was calculated by a non-conditional logistic regression. Significant differences in categorical variables among the parameters were confirmed through analytical statistical tests. The optimal threshold with high specificity and sensitivity for PCOS phenotypes was detected using receiver operating characteristic (ROC) analysis. we relied on IBM's SPSS Statistics 28.0 (Chicago, IL, USA) .

RESULTS

The total number of females PCOS 140 : 69(49.28%) phenotype A , 20(14.28%) phenotype B,23(16.42%) phenotype C and 28(20%) phenotype D ,table (2) represent the demographic, laboratory, and metabolic indices of the study group's population. The age group range (18-44years),where the BMI ranges from (19.4-41.6). The analysis data for the hirsutism score (modified Ferriman-Gallwey) was shown that about (75%) of the participants were having hirsutism, Also the present and absent of acne were estimated to be 57.85%, and 42.14% respectively, As well as alopecia was present in 83.57% and while absent in 16.42%. The analysis of ovulatory dysfunction was revealed that about 117 of the PCOS group were have oligo-amenorrhea that represents groups (A, B, and D) of patients while 23 have regular mens group (C). The investigation of Ovarian morphology was shown that 120 have PCO in groups (A, C, and D) of patients and only 20 participants were having normal morphology as in group (B).

Table (2): The demographic characteristics, laboratory, and metabolic measurements in PCOS phenotypes and control.

	Phenotype A (N=69) (Mean±SD)	Phenotype B (N=20) (Mean±SD)	Phenotype C (N=23) (Mean±SD)	Phenotype D (N=28) (Mean±SD)	Control (N=70) (Mean±SD)	P-value
Age(years)	**24.52±5.06**	**24.6±6.34**	**25.17±3.9**	**24.64±4.1**	**27.60±4.10**	**0.052**
BMI(Kg/m^2)	**30.57±6.07**	**27.48±4.53**	**27.97±3.8**	**26.96±4.2**	**23.64±1.96**	**<0.001**
WHR	**0.85±0.05**	**0.85±0.04**	**0.85±0.05**	**0.83±0.05**	**0.79±0.05**	**0.001**
LH(m.lu/mL)	**11.48±3.58**	**10.76±4.19**	**8.63±2.2**	**4.9±1.26**	**4.40±1.15**	**<0.001**
FSH(m.lu/mL)	**6 ± 1.78**	**5.57±1.20**	**5.12±1.12**	**5.82±1.2**	**8.64±1.53**	**<0.001**
LH/FSH	**2.08 ±0.71**	**2.13±0.77**	**1.74±0.54**	**0.85±0.26**	**0.51±0.12**	**<0.001**
TSH(ulU/mL)	**2.33±0.68**	**2.35±0.83**	**2.15±0.74**	**2±0.94**	**2.30±0.48**	**0.16**
Pro(ng/mL)	**18.37 ±6.41**	**14.65±5.69**	**14.7±6.9**	**19.35±6.7**	**15.81±5.53**	**0.06**
F.T(pg/ mL)	**2.47±1.3**	**1.96±1.03**	**2.09±0.86**	**1.91 ±1.08**	**0.83±0.44**	**0.003**
HOMA-IR	**3.57±0.75**	**2.7±0.7**	**3.3±0.93**	**2.3±0.94**	**1.38±0.33**	**<0.001**
LDL(mg/dl)	**95.90±26.07**	**78.43±32.7**	**83.09±17.02**	**82.68±26.5**	**70.59±16.31**	**<0.001**

HDL(mg/dl)	55.84 ±6.78	51.4±8.15	54.8±7.17	55.5±6.6	65.83±4.86	<0.001
TG(mg/dl)	96.38 ±27.03	78.96±30.3	81.6±27.4	71.06±23.2	74.43± 15.13	0.001
TC(mg/dl)	163.47 ±28.58	142.5±32.2	157.47±19.28	158.42±34.26	156.41±22.44	0.05
VAI	1.34 ±0.5	1.07±0.4	1.13±0.45	0.91±0.4	0.81 ± 0.21	<0.001
LAP(cm.mmol/L)	27.52 ±16.21	20.75±10.8	23.19±14.78	16.8±10.65	17.45 ± 11.74	<0.001
BAI%	35.26 ±7.07	32.4±5.26	33.15±4.49	31.39±7.61	29.79 ± 6.37	<0.001
BMR	1531.49±149.7	1438.35±141.26	1441.81±97.24	1441.2±123.8	1412.67±106.16	<0.001

Table Note: Data expressed as mean ± standard deviation (SD); BAI: body adiposity index; BMI: body mass index; BMR: basal metabolic rate; WHR: waist hips ratio; F.T: free testosterone; FSH: follicle-stimulating hormone; LAP: lipid accumulation product; LH: luteinizing hormone; LDL: low-density lipoprotein; HDL: high-density lipoprotein; HOMA-IR: homeostasis model assessment of insulin resistance; Pro: Prolactin; TSH: Thyroid-stimulating hormone; TC: Total cholesterol and VAI: Visceral Adiposity Index.

The association of the VAI, LAP, BMR, and BAI with the PCOS phenotype group. It was found that all the parameters were highly significant differences in PCOS phenotypes group , while only BMR was significant in group A. VAI consider a fifteen-time risk in phenotype A than in phenotype D. LAP ,BMR, and BAI were highly risk in phenotype A as shown in table(3).

Table (3): The multinomial logistic regression of PCOS phenotype groups with metabolic measurements

Variable	Groups	OR (Lower – upper)	P value
VAI			
	Phenotype A	45.99 (12.99-162.77)	<0.001[S]
	Phenotype B	12.40 (2.71-56.67)	<0.001[S]
	Phenotype C	18.13 (4.28-76.75)	<0.001[S]
	Phenotype D	3.64 (0.80-16.51)	0.005[S]
	Control Group	1[a]	-
LAP			
	Phenotype A	1.05 (1.02-1.09)	<0.001[S]
	Phenotype B	1.02 (0.98-1.07)	<0.001[S]
	Phenotype C	1.04 (1.00-1.08)	<0.001[S]
	Phenotype D	0.99 (0.95-1.03)	0.07[NS]
	Control Group	1[a]	-
BMR			
	Phenotype A	1.007(1.004-1.010)	0.003[S]
	Phenotype B	1.002 (0.998-1.006)	0.356[NS]
	Phenotype C	1.002 (0.998-1.006)	0.273[NS]
	Phenotype D	1.002 (0.998-1.006)	0.250[NS]
	Control Group	1[a]	-
BAI			
	Phenotype A	1.14 (1.07-1.21)	<0.001[S]
	Phenotype B	1.07 (.99-1.16)	0.008[S]
	Phenotype C	1.09 (1.01-1.18)	<0.002[S]
	Phenotype D	1.04 (0.97-1.12)	0.05[S]
	Control Group	1[a]	-

p<0.05 considered significantly different, [S]= Significant, [NS]= Non significant

1[a]: reference category is Control

ROC curve and AUC analysis for the metabolic indices in PCOS Phenotypes in table (4) .The AUC was extremely significant p-values (< 0.05). Results of the Sensitivity & Specificity were confirmed using Youden's J statistics.

Table(4): Receiver operating characteristic curve showing sensitivity and specificity of metabolic measurements in PCOS phenotypes

Variable	Phenotypes	AUC	P value	Sensitivity %	Specificity %	Cut off	Youden index	CI %
VAI	A	0.85	<0.001	71%	89.7%	1.045	0.60	0.78-0.92
	B	0.73	0.003	70%	75%	0.81	0.45	0.59-0.88
	C	0.77	<0.001	69.6%	85%	0.91	0.54	0.64-0.9
	D	0.61	0.116	57.1%	75%	0.80	0.32	0.46-0.76
LAP	A	0.80	<0.001	79.7%	74.4%	15.72	0.54	0.71-0.89
	B	0.71	0.008	80%	70%	13.97	0.5	0.55-0.86
	C	0.74	0.001	82.6%	70%	14.11	0.52	0.60-0.87
	D	0.59	0.178	75%	50%	9.85	0.25	0.45-0.73
BAI	A	0.84	<0.001	76.8%	89.7%	30.01	0.66	0.77-0.92
	B	0.80	<0.001	75%	85%	29.69	0.6	0.68-0.92
	C	0.85	<0.001	78.3%	90%	29.9	0.68	0.76-0.95
	D	0.71	0.003	57.1%	85%	29.6	0.42	0.58-0.84
BMR	A	0.83	<0.001	81.2%	76.9%	1420.21	0.58	0.75-0.90
	B	0.61	0.158	35%	99%	1553.33	0.34	0.44-0.77
	C	0.67	0.019	65.2%	70%	1408.23	0.35	0.53-0.81
	D	0.63	0.062	46.4%	90%	1456.54	0.36	0.49-0.77

DISCUSSION

Polycystic ovarian syndrome the diagnostic features, ovulatory dysfunction (oligo/ amenorrhea) ,clinical and/or biochemical hyperandrogenism , or polycystic ovaries morphology . Hyperandrogenism is also associated with IR ,due to insulin having a gonadotropin-stimulating effect, which lead to the defining characteristic of PCOS's metabolic and reproductive processes. Insulin not only increases adrenal and ovarian steroid production, but it also increases pituitary LH secretion (**23**). Early detection of IR and treatment to improve insulin sensitivity in PCOS patients is crucial. Recent attempts to apply new metabolic measurements such as the VAI, LAP, and BAI, display a high degree of accuracy in the detection of abdominal obesity. In addition, research indicates that they might be accurate indicators of IR, metabolic syndrome (MS), T2DM, and cardiovascular in PCOS (**23-25**).

Recent research has shown that the VAI, LAP, and BAI are predict insulin resistance (IR) better than BMI and other classic measures, evaluating diagnostic accuracy, and comparison of numerous adiposity, cardiometabolic, and insulin resistance indices to determine the most accurate predictor of insulin resistance and MS risk in polycystic ovary syndrome women of reproductive age because they take into account the etiology and anatomical modifications brought on by the accumulation of fat (**8**). Asian populations are more likely to manifest fat accumulation and insulin resistance than Western populations (**26**). The androgen production and metabolic clearance of PCOS patients with visceral adiposity are modified. androgen increases promote lipolysis and enhance the outflow of free fatty acids and IR. Visceral obesity has an important effect on androgen and IR metabolism (**23**) .

Observed the result of phenotype A most significant and high risk in association, Phenotype C, phenotype B, and less phenotype D in VAI shown in Table (3), result increase in HOMA-IR in phenotype A the classic one phenotype C ,phenotype B and phenotype D as shown in table (2).The study by Bil et al in 2016 the most significant was Phenotype B (**27**). Variations in the ethnicity, age, BMI, and dietary patterns of study participants may account for VAI threshold differences among various study (**28**). The phenotype A shown that AUC is 0.85 with cut-off value of 1.045(Sensitivity71 % and specificity 89.7%).

In this study funding for LAP is significant in Phenotypes (A, B, and C), with the more significant risk in Phenotype A as shown in Table (3). previse study by Bozic-Antic et al in 2016 that Phenotype B (**29**). This difference as being due to ethnic composition. The AUC is 0.80 with a cut-off value of 15.72(Sensitivity 79.9 % and Specificity 74.4%).

This study, the funding that all PCOS Phenotypes is significant in BAI, while Phenotype A is riskier and more significant than the other Phenotypes. There is no research published on the BAI in different phenotypes, While the research in the PCOS patients' group and control considered the BAI a good indicator in the detection of cardiovascular risk and insulin resistance in PCOS. (**14**,**30**). In the investigation by Johnson et al BAI was found to be far better than BMI in predicting adult adiposity, BMI does not distinguish between fat and muscles and it was suggested that it is the most accurate predictor of insulin resistance (**31**)

In 2020 by Yesil et al that BAI is not a much better index than Bioelectric Impedance Analysis (BIA) and skinfold thickness in determining body fat %, the BAI is a straightforward method for estimating the amount of fat in a person's body without measuring their weight. Without more complex or costly methodologies, such as a measurement of the thickness of the skin or BIA, BAI may serve as an alternative predictor of body fat (**32**).

This study found phenotype A is more significant in VAI, LAP, and BAI more accurate of IR, MS, T2DM, and cardiovascular in PCOS due to it being the classic Phenotype with a high risk of obesity, T2DM, IR, coronary heart disease, and other metabolic disorders (**5**).

Women with PCOS are more likely to be obese, and this has been linked to a variety of adverse metabolic, cardiovascular, endocrine, reproductive, and mental health consequences. When examining associations between nutrients and health, it is essential to accurately measure diet and energy intake (**33**). Patients with polycystic ovary syndrome may also experience difficulties because of their weight. Reducing one's body fat has been demonstrated to improve one's fertility and metabolic profile (**34**).

Patients with PCOS who are obese have been recommended a calorie-restricted diet; nevertheless, weight maintenance is challenging as the majority of participants regain weight, placing them at risk for weight cycling. (**35**).

while an increase in the PCOS patients group compared to the control group in table (2) To our knowledge, BMR in women with PCOS has not been extensively studied these results are supported by the study De Giuseppe et al in 2018 resulting Although PCOS patients had a significantly higher mean BMR than controls(1658.7 ± 201.1 kcal vs 1359.2 ± 103.7 kcal; $P < 0.0001$, respectively) (**36**). In 2009 reported that decrease in BMR in PCOS females(**37**).

It is a risk in all phenotypes while the high risk and more significant in phenotype A. A study by Ilic et al in 2015 resulted in the phenotype (B and C) having higher BMR, their findings demonstrated a correlation between body composition and androgenic status. Consequently, phenotype B exhibited the most altered structure of body composition (**38**),the Variations in this research measured BMR by bioelectrical impedance in our study by the Mifflin-St Jeor equation. Consequently, based on the findings of this study, women with PCOS consume less energy and should reduce their caloric intake to maintain normal body weight.

In this study result, that all PCOS phenotypes significant increase compared to the control group with a p-value < 0.05 .All new metabolic parameters indicate the risk of PCOS in predicting insulin resistance and visceral obesity associated with PCOS. These results are supported by the study conducted Davut Sakiz et al in 2022 that anthropometric measures may be useful in predicting the development of subclinical atherosclerosis and IR in females with PCOS. Similar results were obtained by the study conducted by Marzena Jabczyk et al in 2023 the majority of the analyzed anthropometric indices may be useful in evaluating metabolic disorders, particularly glucose and insulin abnormalities in women with PCOS (**30,39**). The new knowledge added by this study is by dividing the patients into

different four phenotypes groups that hyperandrogenic phenotypes the most risk of insulin resistance and metabolic disturbances. limitation of this study sample size and geographical region in one area that needs in a different area. This study did not assess the cardiovascular indices and new metabolic measurements in different PCOS phenotypes, thus, future research.

CONCLUSIONS

Our investigation of basal metabolic rate and metabolic indices in the PCOS phenotype revealed a statistically significant increase difference between the four phenotype groups studied. The females with phenotype A the classic one have a higher basal metabolic rate and metabolic Measurements than the control group. Consequently, insulin resistance and metabolic disturbances more severe in phenotype A than in other phenotypes.

Declaration of patient consent: All necessary consent forms from patients have been received, as attested by the authors.

Author's contribution: The work's conception and design owe a great deal to everyone's contributions. All authors approved the final version for publication and agreed to be responsible for all parts of the work, including conducting adequate research to resolve any concerns about the accuracy or integrity of any portion of the work.

Financial support and sponsorship: Nil.

Conflicts of interest: There are no conflicts of interest.

Abbreviations:

AUC Area Under Curve

BAI body adiposity index

BMI Body mass index

BMR basal metabolic rate

HA hyperandrogenism

IR insulin resistance

LAP lipid accumulation product

OD ovulatory dysfunction

PCOM polycystic ovaries morphology

PCOS polycystic ovarian syndrome

T2DM Type 2 Diabetes Mellitus

VAI visceral adiposity index

WHR Waist Hip Ratio

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